Carotid artery aneurysm: Evolution of management over two decades

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Introduction: **Extracranial carotid artery aneurysm (CCA), although uncommon, represents a challenge to treatment strategy. The purpose of this study was to analyze the treatment evolution and clinical outcome of all patients with CCA over a two decade period.**
Methods: **Clinical data of all patients diagnosed with CCA who underwent interventions from 1984 to 2004 were reviewed. Patients were divided into two groups. Group I (1985-1994) and group II (1995-2004) were compared with regards to clinical presentation, treatment modality, and clinical outcome.**
Results: **A total of 42 cases of CCA were found during the study period (group I, n=22; group II, n=20). Pulsatile neck mass was the most common presenting symptom (n=39, 93%), followed by neurological symptoms (n=6, 14%). Twenty two (52%) were atherosclerotic aneurysms, fifteen (36%) false aneurysms, and five (12%) posttraumatic aneurysms. Both groups shared similar comorbidities and demographic profiles. All patients in group I underwent operative interventions, which included 12 resection with interposition bypass grafting (55%), six resection with patch angioplasty (27%), and four carotid ligation (18%). In group II, five patients underwent resection with interposition placement (25%) and one carotid ligation (5%). The remaining 14 patients underwent endovascular interventions (70%) which included seven stent-graft exclusions, six carotid stenting with coil exclusions, and one endovascular occlusion. Hospital length of stay was significantly shorter in group II than group I (3.5 vs. 9.4 days, $p<0.01$). The incidence of cranial nerve injury in group I and II were 14% vs. 5% ($p<0.04$), respectively. The 30-day mortality/major stroke rates in group I and II were 14% vs. 5% ($p< 0.04$), respectively. During the follow-up period (0.8 months-20 years; mean, 4.6 years), 16 patients died, largely due to cardiac etiologies (n=11, 69%).**
Conclusions: **Treatment modality of CCA has largely evolved from operative to endovascular intervention at our institution. Treatment benefits of endovascular modality include shorter convalescent and less procedural-related complications. This evolution reflects the improvement of endovascular devices and increased utility of endovascular applications. (J Vasc Surg 2006;43:493-6.)**

INTRODUCTION

Aneurysm of the carotid artery aneurysm is an uncommon clinical disease which can occur as the result of atherosclerotic degeneration, fibromuscular dysplasia, or traumatic injury. Infrequently, this can also occur as a complication following a carotid endarterectomy (CEA) procedure. Because of the risk of aneurysm rupture as well as the neurological sequalae caused by cerebral atheroembolism, surgical intervention is recommended for a carotid artery aneurysm (CCA) in order to minimize potential complications.

A variety of operative techniques have been described in the treatment of CCA. Sir Astley Cooper reported successful treatment of CAA by proximal carotid artery ligation in 1808.[1] Subsequently, Dimtza performed first successful resection and re-establish the continuity of carotid artery in 1956.[2] In the past four decades, most surgeons have adapted the surgical technique of aneurysm resection with either interposition grafting or patch angioplasty as the operative treatment of choice. Despite this well-recognized treatment technique, operative repair of CCA often represents a significant challenge to surgeons particularly in the presence of extensive inflammation in the surrounding tissues caused by the aneurysm. Moreover, the internal carotid artery dissection for distal control can pose added technical challenges when a CCA extends well above the carotid bifurcation.

Recent advances in endovascular therapy have resulted in various minimally-invasive treatment modalities in arterial aneurysmal diseases. Numerous clinical studies have documented the efficacy and safety of utilizing endovascular techniques including stent-graft exclusion or coil embolization in the management of arterial aneurysms.[3-5] We have also witnessed an increased utilization of endovascular modalities in the management of arterial aneurysms, including the CCA, in our clinical practice. The increased treatment options of CCA, which include both open and endovascular interventions prompted our interest to review our experience in the treatment of this disease. In this article, we report our experience in the management of CCA over the last two decades. Specifically, we described the changing trend of our treatment strategies and analyzed

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the clinical outcome in patients following either open or endovascular interventions.

MATERIALS AND METHODS

From September 1985 to October 2004, all patients who underwent operative repairs for arterial aneurysms (n=7,234) were identified by current procedural terminology (CPT) codes at the Baylor College of Medicine-affiliated hospitals, which included the Michael E. DeBakey VA Medical Center, the Ben Taub General Hospital, and the Methodist Hospital. Among these patients, 42 patients (0.6%) underwent operative repair of extracranial carotid artery aneurysm (CCA) which formed the basis of our patient cohort. In addition to demographic information, data were obtained on patient's relevant co-morbidities, preoperative imaging studies, treatment indication, therapeutic modality, and clinical outcome. Particular attention was paid to the treatment modality with regards to operative or endovascular approach. Long-term outcome was assessed by means of duplex ultrasound and clinic visit records. For the purpose of comparison, patients were divided into two groups based on the treatment period. Group I consisted of patients treated during the first 10 years of the study period, while group II included those treated the latter 10 years. Analysis was made with regards to the treatment modality and clinical outcome between the two groups.

Statistical analyses were performed by using the pooled Student *t* test and uncorrected Pearson chi-square test. A *P* value less than .05 was considered to be statistically significant. These calculations were performed using the Statistical Package of Social Sciences for Windows (SPSS Inc., Chicago, IL). P values of <=0.05 were considered statistically significant.

RESULTS

Demographic data

During the study period, a total of 42 cases of CCA repair were performed. Among them, 22 patients underwent operative repair from 1985 to 1994 (group I) while 20 patients underwent either open or endovascular interventions for CCA from 1995 to 2004 (group II). In group I, 86% of the patients were male (n=19), similar to 85% in group II (n=17). The age range for the entire patient group was 22 to 87 years. The mean age was 54 ± 16 years in group I versus 58 ± 18 years in group II. Demographic variables of the two groups are listed in Table I, which were similar in terms of their associated comorbidities.

Indications for intervention

Underlying etiologies of CCA are listed in table I, which were categorized as either post-carotid endarterectomy pseudoaneurysm, atherosclerotic aneurysm, or traumatic aneurysm. Among our patients, 22 (52%) were atherosclerotic aneurysms, 15 (36%) pseudoaneurysms, and 5 (12%) posttraumatic aneurysms. Presenting symptoms associated with CCA are also summarized in Table I. Pulsatile neck mass was the most common presenting symptom (n=39, 93%), followed by neurological symptoms (n=6, 14%). Specifically, symptoms associated with neurological deficits included transient ischemic attack (n=2), stroke (n=3), and Horner syndrome (n=1). There was no difference in clinical presentations or underlying etiologies between the two groups.

Table I. Patient demographics and clinical characteristics

	Group I, N=22 [n (%)]	*Group II, N=20 [n (%)]*	P *value*
Patient characteristics			
Age (mean, years)	54 ± 16	58 ± 18	NS
Sex (Male)	19 (86%)	17 (20%)	NS
Comorbidities			
Coronary artery disease	16 (73%)	15 (75%)	NS
COPD	5 (23%)	7 (35%)	NS
Hypertension	18 (82%)	17 (85%)	NS
Diabetes	3 (14%)	2 (10%)	NS
Chronic renal insufficiency	2 (9%)	2 (10%)	NS
Etiologies			
Atherosclerotic degeneration	12 (55%)	10 (50%)	NS
Pseudoaneurysm	8 (36%)	7 (35%)	NS
Post-traumatic aneurysm	2 (9%)	3 (15%)	NS
Presenting symptoms			
Pulsatile neck mass	20 (91%)	19 (95%)	NS
TIA	1 (5%)	1 (5%)	NS
Stroke	1 (5%)	2 (10%)	NS
Horner's syndrome	1 (5%)	0	NS

Table II. Treatment strategies

	Group I n (%)	*Group II n (%)*
Operative repair		
Resection and interposition grafts	12 (55%)	5 (25%)
Resection and patch angioplasty	6 (27%)	0
Carotid ligation	4 (18%)	1 (5%)
Endovascular repair		
Stent graft exclusion	0	7 (35%)
Stent placement with coil exclusion	0	6 (30%)
Endovascular balloon exclusion	0	1 (5%)
Total	22 (100%)	20 (100%)

Types of interventions

Treatment strategies are listed in Table II. Briefly, all patients in group I underwent operative repair, which included 12 resection with interposition bypass grafting (55%), six resection with patch angioplasty (27%), and four carotid ligation (18%). In contrast, patients in group II underwent either open or endovascular treatment. Among them, five patients who were treated resection and interposition graft placement (25%), one received carotid ligation (5%) and the remaining 14 patients were treated with endovascular methods (70%), which included stent-graft exclusions (n=7, 35%), carotid stenting with coil exclusions (n=6, 30%), and endovascular balloon occlusion (n=1, 5%). In group II, All patients received life-long

Table III. Clinical outcomes

	Group I, N=22 [n (%)]	*Group II, N=20 [n (%)]*	P *value*
Technical success	22 (100%)	20 (100%)	NS
Cranial nerve injury	3 (14%)	1 (5%)	<0.04
Wound infection/ complication	2 (9%)	0	<0.05
Length of hospital stay (days)	9.4 ± 3.5	3.5 ± 1.2	<0.01
30-day stroke rate	1 (5%)	0	NS
Perioperative death rate	2 (9%)	1 (5%)	NS
30-day stroke and death rate	3 (14%)	1 (5%)	<0.04

aspirin therapy and patients undergoing endovascular interventions received an additional six weeks of clopedigrol (75 mg per day).

Treatment outcome

Clinical outcomes are summarized in Table III. Technical success was achieved in all patients, including aneurysm resections in surgically treated patients and aneurysm exclusions in patients treated with endovascular methods. Three patients (14%) in group I suffered from cranial nerve injuries following operative repair, in contrast to one patient (5%) in group II ($p < 0.04$). Wound complications occurred in two patients (9%) in group I while no patients in group II developed wound-related complications ($p < 0.05$). Patients in group II had a significantly shorter length of hospital stay than group I (3.5 ± 1.2 days vs. 9.4 ± 3.5 days, $p<0.01$). No significant differences in either 30-day stroke rate or perioperative death rate were noted when comparing the two groups. However, the combined 30-day stroke and death rate was higher in group I (14%) when compared to group II (5%, $p<0.04$). During a mean follow-up period of 4.6 year (range, 24 days-20 years), 16 patients died, of which, 11deaths were due to cardiac etiologies (69%) and procedural-related mortality were similar between group I and group II (14% and 10%, respectively).

DISCUSSION

Carotid artery aneurysms, albeit rare, pose treatment challenges due to formidable surgical complications and frequently inaccessible locations. With the rapid advancement of endovascular techniques, therapeutic strategy has shifted toward catheter-based intervention from traditional open repair. Our series demonstrated the evolution in treatment strategies of CAA over the last two decades in our institution and highlights the endovascular therapy as a feasible and durable alternative with the benefits of shorter convalescence and less procedure-related complications. This evolution reflects the improvement of endovascular devices and increased utility of endovascular applications.

CAA is only encountered in 0.2-5% of all carotid artery surgeries.[6-8] The most common causes of CAA are atherosclerotic degeneration, fibromuscular dysplasia, trauma, and CEA-related complications. Studies have demonstrated the incidence of CAA ranging from 0.02% to 0.4% among all trauma patients. Hughes et al identified seven patients with CAA among 3342 trauma patients over a three-year period and among 189 patients undergoing MRI screening.[9] Of the 38 cases reported by Pulli et al, 26% were caused by atherosclerotic degeneration, 16% were due to dysplastic changes, and 13% were pseudoaneurysms. Their findings were also confirmed in an earlier report from our institution by McCollum et al who reviewed 37 extracranial CAA over 21 years and found that 44% were atherosclerotic aneurysms, 51% were pseudoaneurysms, and 5% were due to trauma.[10] Furthermore, another large series of 67 cases reported by El-Sabrout and Cooley demonstrated that 57% were resulted from prior CEA and 43% were secondary to atherosclerosis or trauma.[11] Our series of 42 patients demonstrated similar distribution in etiologies of CAA as reported in the literature. 50% of our patients had atherosclerotic aneurysms, 38% had false aneurysms, and 12% were caused by trauma.

The natural history of CAA is conical growth and thus, has the potential for rupture, distal embolization, and local compression. Neurological symptoms are common in this group of patients. McCollum et al identified 15% of their patients had neurological symptoms and palpable masses in all patients.[10] In El-Sabrout report, 59% of patients presented with cervical masses, 43% had neurological symptoms, more than 10% had local compression symptoms, and 3% of patients presented with rupture or impending rupture.[11] Moreau and colleagues also confirmed 74% of their patients had cerebral ischemic symptoms.[12] Similar to the reports in the literature, 14% of our patients also had neurological deficits.

The potential severe complications associated with CAA obligate surgical intervention. A review by Winslow indicated that nonoperative management had mortality rate of 71%, while carotid ligation was associated with 28% mortality.[13] El-Sabrout and Cooley reported mortality and major stroke rate of 9%-12% for surgical reconstruction as comparing to 50% mortality after ligation. Surgical treatment generally involves five options including aneurysm clipping, resection and end-to-end anastomosis, resection with interposition graft, extracranial to intracranial bypass, and carotid artery ligation. Despite decreased complication rate after open surgical repair over time, the morbidity is still significant. Moreau and associates reported only 2.6% perioperative mortality, yet 26% of all surgically treated patients had cranial nerve palsy.[12] In 2000, Hertzer addressed extracranial carotid aneurysms in a commentary and indicated that although rate of stroke is only 4% in his experience, the incidence of cranial nerve dysfunction approaching 44%. He further raised the questions on possibly medical management of symptomatic pseudoaneurysms due to high mortality and morbidity. Our open surgical treatment included resection and interposition grafting, resection and patch angioplasty, and ligation. All three cranial nerve injuries in our patients occurred during open surgical repairs. Perioperative mortality and major stroke rate in the open surgical group between 1985 and 1994 was

12% comparing to 5% in group II, in which 65% of the patients underwent endovascular interventions.

Endovascular approach offers the advantage of avoiding difficult dissection and eliminating the needs for high cervical exposure, thus reducing the risk of cranial nerve injuries and other procedure-related complications. Several endovascular techniques have been used to treat CAA, including bare stent with or without transstent coiling,[4,5] autogenous vein covered stents,[14] endovascular balloon occlusion,[15] and stent-grafts, such as Viabahn® (WL Gore, Flagstaff, AZ) and Wallgraft® (Boston Scientific, Natick, MA).[16,17] Our group, in 2001, evaluated 5 patients received endovascular interventions for carotid artery pseudoaneurysms and reported 100% patency rate during an average 8.4 months follow up, ranging from 2 to 21 months.[4] Most endovascular treatments of CAA are derived from experience of managing traumatic or intracranial pseudoaneurysms. Saatci et al reported using stentgraft treating 25 distal ICA aneurysms and majority of the aneurysm were post-traumatic.[3] They reported no aneurysm recanalization during their six months to two year follow-up and all initial symptoms due to mass effect were resolved. Additionally, Coldwell and colleagues treated 14 patients with traumatic pseudoaneurysms using primarily Wallstents with additional Palmaz stent in selected patients.[5] During a mean follow-up of 16 months, no patient had worsening of their neurological status or complications attributable to the presence of the Wallstent. Our series demonstrated similar results. During a mean follow-up period of 4.6 year, eleven of the 16 deaths were due to cardiac etiologies and continued aneurysm exclusion was confirmed in all patients.

This review highlights an evolution in the treatment of CAA at our institution. Patients treated before 1995 were exclusively repaired with open surgical interventions while later patients were treated with either surgical or endovascular therapies depending on the individual surgeon's preference and the locations of the lesions. The patients with lesions in distal cervical locations tended to be treated with endovascular methods. Within the limitations of an observational data set, our series clearly demonstrates that endovascular intervention is a feasible and durable alternative with the advantages of less convalescence and procedure-related complications. Despite isolated reports of complications related to endovascular intervention,[18] it is a safe and effective therapeutic option in experienced hands. However, long-term follow-up is warranted.

In conclusion, our experience of treating CAA over the last two decades reflects the evolution of treatment modalities. Our review demonstrated endovascular intervention as an effective alternative in treating CAA with less procedure-related complications and shorter recovery time particular for patients with prohibitive surgical risks or patients with distal located aneurysms that preclude safe surgical approach.

AUTHOR CONTRIBUTIONS

Conception and design: W.Z, P.H.L, R.L.B
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